

REPORT ON PATHOLOGY AND PATHOLOGICAL ANATOMY.

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GENERAL PATHOLOGY.

Fungi in the Blood.—Riess (*Centrbl.*, 1873, p. 530) takes exception to some of the statements of Birch-Hirschfeld (previously referred to in this JOURNAL), and, after a series of investigations, comes to the conclusion that there are no means, chemical or microscopical, by which micrococci can be distinguished in the animal organism from detritus. In consequence, the assigned importance of the many abnormal elements found in the blood and organs, in infectious diseases, is not possible, as no sure proof of the parasitic nature of such processes has been obtained.

Birch-Hirschfeld (*Centrbl.*, 1873, p. 609), in reply, refers to his published recognition of the detritus referred to by Riess, but still retains his opinion that, in addition, globular bacteria are present, and that their exact nature is to be determined, in part, through chemical reagents. He admits, at the same time, that the parasitic nature of such diseases is not yet assured.

Obermeier (*Centrbl.*, 1873, p. 145) has discovered, in the blood of individuals suffering from recurrent fever, extremely delicate, thread-like bodies, of the thickness of a fine thread of fibrine, and as long as the diameters of from $1\frac{1}{2}$ to 6 or more blood corpuscles. The blood being removed from the patient and the corpuscles allowed to become quiet, it was found that these threads possessed extremely rapid movements. These movements are of two kinds, undulating and locomotive. When locomotion took place, the threads became crooked, circular or corkscrew like; then elongating, they would leave the field of vision either gradually or suddenly. Locomotion ceases after one to two hours; undulation may continue up to eight hours. They are easily to be distinguished from threads of fibrine. These bodies were first seen by their discoverer in 1868, but lack of material has hitherto prevented investigation. Up to the time of publication, they had been found only during the fever, and shortly before or during the crisis, not in the remission. No decision was arrived at as to their nature.

Obermeier (*Centrbl.*, 1873, p. 561) inoculated animals with the blood from patients with recurrent and typhoid fever. The blood was injected subcutaneously, and into the jugular veins of dogs, rabbits and guinea-pigs. The results were negative. Nevertheless, he considers that the presence of the contagium in the blood is not thereby eliminated. Accidental or intentional inoculation of such blood, through cracks in the epidermis, produces no infection. As an explanation for the lack of success in producing the disease experimentally, he suggests that special conditions or preparations are necessary for the reception of the contagium in the living organism.

Wolff (*Centrbl.*, 1873, p. 497) has continued his experiments with regard to the effect of fungi introduced into the blood. It may be remembered that he was unable to conclude that the active deleterious principle of putrid blood was to be found in bacteria. In his present

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publication, he gives the results of experiments with the contents of metastatic abscesses and the secretion from gangrenous wounds. In comparing the action of these fluids with that of a fluid in which fungi from the preceding were cultivated, he found that the latter, in the same doses, was by far less deleterious than the former. With the microscope, the same forms of fungi were found in the three fluids. The bacteria were of various forms, a noteworthy observation where special forms of bacteria have been regarded as productive of special diseased processes. In addition, he records that, with Hartnack 10, prismatic bodies were found in the peritoneal exudation of puerperal fever, hardly as large as the diameter of a red blood corpuscle.

Küssner (*Centrbl.*, 1873, p. 497) opposes the view that the microsporon septicum is characteristic of the septic processes, as he finds the same in trivial abscesses. He states that he was able to free fluids from bacteria by filtering through a double layer of filter paper in a glass funnel, whose nozzle was plugged with boiled cotton-wool. The filtrate, as well as the fluids containing bacteria, produced death after injection; the *post-mortem* appearances were alike—ichorous and purulent infiltration of the cellular tissue, and, finally, abscesses at the point of injection. There was no pathological alteration of the internal organs, nor were bacteria found in the blood, nor in the greater glands of the abdomen. He concludes that the action of the injected fluids depends upon chemical processes of decomposition, independent of the bacteria. A temperature curve, characteristic of sepsin poisoning (Tiegel), was not found.

Inoculability of Cancer.—Desirous of ascertaining the effects of cancer grafts upon the body of rabbits, Hyvertl (*Gaz. des Hôp.*, No. 49, 1873, *Allg. Med. Centr. Zeit.*, 1873, p. 602) undertook a series of experiments. The portions of tumor free from pus, and not far advanced in development, were introduced deep into the tissues as far as the muscular layer.

Inflammation and induration resulted, the latter to be felt five months afterwards as a "tumor." This was surrounded by a tissue rich in vessels, and presented its previous appearance: cancer cells and alveoli at the periphery, granulations commenced towards the centre; at the latter point was calcification. The autopsy showed that neither infection nor general disease resulted. The author concludes that young cancer cells, transplanted from man to animals, do not produce cancer.

Exophthalmic Goitre.—Boddaert (*Bull. de la Soc. de Méd. de Gand, Gaz. Méd.*, 1873, p. 141) experimented on rabbits with reference to the origin of this condition. Ligatures were placed upon the external and internal jugular veins at the base of the neck, and the two cervical cords of the sympathetic were cut. An exophthalmia resulted, continuing several days, diminishing gradually as the collateral venous circulation became developed, and as the effects of the section of the sympathetic disappeared. Exophthalmia following the ligature alone, due to distention of the orbital veins, is much less pronounced. An enlargement of the thyroid is produced by section of the sympathetic and ligature of the inferior thyroid vein between the four jugulars. These experiments, combined with the discovery of lesions of the sympathetic, whose effects are analogous to those produced by section (atrophy of nerve elements, hypertrophy of connective tissue)

in a number of cases of Basedow's disease, are considered as explaining the phenomena of the disease. In exophthalmic goitre, an obstruction to the circulation occurs; the superficial veins, especially of the neck, become swollen; there is a tendency to hæmorrhage, an increase of splenic and hepatic dulness, occasional dropsies, œdema, and the enlargement of the retinal vessels observed by Graefe. Boddaert hence produces this theory of exophthalmic goitre. In the majority of cases, the pulsations of the heart increase in number—120 to 200 even; this may continue for months. The veins are insufficiently emptied during the diastole; a venous congestion results, more marked from a more or less complete paralysis of the sympathetic. The effects become most marked in the eye and thyroid body, from the development of the retro-ocular venous system and the great vascularity of the thyroid. This theory is considered as explaining the observation of Trousseau, where the exophthalmia and the thyroid tumor came on during a night, the goitre disappearing suddenly and returning afterwards; also, the diminution of the exophthalmia and the thyroid body, as the heart beats less rapidly.

SPECIAL PATHOLOGICAL ANATOMY.

Ulcerative Endocarditis.—Dr. Lanceraux publishes a paper (*Archiv. Gén.* 1873, p. 672) endeavoring to establish a causal relation between the paludal poison and the ulcerative, vegetative form of endocarditis. He states at the outset that he has already endeavored to prove that rheumatism is not the source of the vegetative, ulcerative endocarditis occurring in the puerperal condition. His ground being that the anatomical characteristics of the rheumatic and puerperal endocarditis differ, hence they cannot have the same origin. Though in both diseases the mitral valve is by preference affected, in the rheumatic form, the entire valvular orifice is diseased, while puerperal endocarditis is always limited and circumscribed. In the former instance, permanent valvular lesions must occur, while, in the latter, a permanent organization of the new tissue is impossible, its destruction producing a fatal infection, so to speak, of the organism. The object of the present paper is to show that there is a vegetative, ulcerative endocarditis, preferring the aortic valves, and common among people who have had intermittent fever. From its localization, anatomical characters and evolution, it must bear a certain relation to the paludal poison.

A number of cases are reported, the clinical phenomena bearing a resemblance to the disturbances produced by septicæmia. In the cases reported, the puerperal condition could not have been a cause, and, as the lesions of rheumatism are regarded as different, this affection must be eliminated from the causes. The symptoms are considered as excluding the latter disease. The pains, existing in certain cases, were of a different character from those occurring in acute articular rheumatism and were attributed to the effect of a general infection. The coincident history of malarial infection in all the cases and the identity of the alterations render it probable or at least worthy of investigation, as a relation of cause and effect. It is possible that alcoholic excess may have aggravated the condition. The disease is distinguished from the chronic aortic affections by the presence of fever. The prognosis is grave on account of the rapid pro-

gress and the tendency to ulceration. The valves once destroyed, a fatal termination results on account of the obstructed circulation and the septicæmia following the destruction. In one of the cases reported, sulphate of quinia produced amelioration.

Brown Induration of the Lungs.—Orth calls attention (*Virch. Arch.* vol. 58, p. 126) to an alteration of the vessels observed in such a case. The capillaries and larger vessels were filled with pigment masses similar to those occurring in the interstitial tissue. The vessels at times appeared as if actually injected with brown, granular pigment. Artificial injection showed that such portions were completely obstructed. He regarded the appearance of certain communicating vessels as indicative of compensation for this obliteration. Such vessels pursued a more direct course, and had but few collateral branches. The previously described condition of the vessels he considers as indicating that a direct formation of pigment, from the red blood corpuscles, may take place without the mediation of other cells (Langhans).

Inflammation of the Lungs.—Lungenentzündung, Tuberkulose und Schwindsucht. L. Buhl, Prof. &c. München, 1872.

Untersuchungen über Lungenentzündung &c. C. Friedländer. Berlin, 1873.

Prof. Buhl's monograph of 164 pages is one demanding the most thorough attention from all interested in such matters. Whether the views are accepted or not, valuable suggestions are presented, and in such a form that the continued interest of the reader is retained.

Within the space allotted to this report, a complete abstract is impossible. The text appears in the form of twelve letters, and includes the various forms of acute and chronic inflammation of the lungs, together with the author's ideas as to tubercle, tuberculosis and tuberculous pneumonia, with a final chapter devoted to phthisis.

The distinction between a superficial and parenchymatous inflammation of the lungs holds good only so far as the one or the other condition predominates, the two always co-existing. The alveolar epithelium is regarded as a continuation of the endothelium of the lymph vessels, expanded over the free surface of the alveolar wall, and not as a continuation of the epithelium of the bronchial tubes. The superficial inflammations are those where the products originate in the epithelium, or flow over its surface; the products of the parenchymatous forms are interstitial and peri-bronchial.

The terms lobular and lobar are applied rather to the method of origin and extension, than to the boundary. Lobar refers to primary disease of the pulmonary parenchyma; lobular, to primary disease of the bronchi. Those diseases proceeding from the lymph vessels are not easily divided into these two forms. The chronic forms are particularly distinguished from the acute by the presence of extensive and important degenerative conditions when superficial; by an increase of connective tissue, the formation of cicatrices, &c.; when parenchymatous, interstitial or peri-bronchial.

The acute catarrhal pneumonia of the books is an acute disease, affecting primarily the posterior portion and edges of the lower lobes, presenting the appearances thus described by Rokitsansky:—"the finer bronchi and their terminations are not unfrequently the seat of an intensive inflammation, which becomes at once extended over the pulmo-

nary tissue; this appears, generally, in numerous lobular portions, swollen, hepatized or filled with pus." The term catarrhal pneumonia demands a catarrh in the pulmonary tissue, and the presence of a mucous membrane; as the latter does not exist, the term is improper. A catarrh of the mucous membrane of the smaller and smallest bronchi, filling these with pus and mucus, even when extending to the alveoli, cannot be a catarrhal pneumonia; the alteration of the latter is modified and secondary. The catarrhal pneumonia is "a capillary bronchitis, a bronchiolitis, in which the lungs participate through collateral œdema, atelectasis, local emphysema and engorgement, as a result of the transfer of the bronchial secretion to the individual alveoli." The œdema is merely a collateral phenomenon of the capillary bronchitis; atelectasis and emphysema are not inflammatory.

The bluish-red color of the atelectatic lobules represent a distention of the capillaries with blood when the intra-alveolar pressure has ceased. The lobular nature of the affection is due to the presence of material in the communicating and adjoining alveoli, produced elsewhere, and transferred through aspiration and migration (pus corpuscles, i. e. white blood corpuscles).

Death or recovery may take place. The latter event is based upon the fact that the true pulmonary parenchyma remains intact. Expectoration, fatty degeneration and absorption permit the removal of the products of inflammation and the access of air.

If these changes do not take place speedily, as in feeble children, the aged and in the course of severe disease, certain portions may remain permanently altered, as local emphysema, and, more particularly, atelectasis, in which latter instance, the parts affected may become converted into pigment nodules or cheesy masses. In addition, the plugs in the smaller bronchi and alveoli may remain permanently as cheesy masses. The alterations, when superficial, produced by foreign bodies, bear a very close resemblance to those occurring in so-called catarrhal pneumonia. A rare termination of the bronchiolitis is the dilatation of the alveoli and bronchi, accompanied with atrophy, thereby distinguished from that resulting from other causes, as peribronchitis. Catarrhal pneumonia is, therefore, a capillary bronchitis.

Croupous pneumonia is primary in the parenchyma, secondary in the bronchi.

The term *desquamative pneumonia* may represent three degrees. The lowest degree appears in severe general diseases as a *consecutive desquamative pneumonia*, under those conditions where parenchymatous alterations of the heart, liver, kidneys, &c., occur, and might, in like manner, be called parenchymatous pneumonia.

It is generally double, diffuse, lobar, affects the bronchi secondarily, and may be found in the anterior and upper parts of the lungs as well as in the posterior and lower portions. If tolerably general and distinct, the lungs are enlarged, filled with blood, with isolated, punctate sub-pleural and parenchymatous extravasations. They are œdematous, and do not collapse, barely even on section. Serum, containing minute air-bubbles, flows from the cut surface, and delicate elevations are presented, the projecting walls of the empty alveoli. The tissue is flaccid and friable. With the microscope, one finds abundant alveolar epithelium, swollen, rounded, finely granular, the granules in part protein, in part fat, the latter rapidly predominating. From the con-

dition of the epithelium, arises the term, such being entirely analogous to the changes occurring in the consecutive parenchymatous nephritis. Pus corpuscles are rarely found; they then arise from the co-incident catarrhal bronchiolitis. Pleurisy is absent, and there are no fibrinous plugs. A speedy or protracted convalescence may occur; likewise death.

In protracted convalescence, a chronic fatty degeneration of the epithelium occurs. Acute atrophy of the lungs may also take place, when "the pleura is not adherent, and there is a complete absence of air in the pulmonary tissue, an unusual laxity, with extensive moisture, a smooth surface on section, a brownish-red color mixed with gray, a more or less dense consistency, cylindrical dilatation and approximation of the acartilaginous bronchi, with livid redness of their mucous membrane."

A special form of parenchymatous pneumonia is that where the exudation follows the course of the interlobular and sub-pleural lymph vessels, distinguished from desquamative pneumonia in that its course is accompanied by pus formation. This process is most frequently seen in pyæmic, new-born infants, infected by the mother suffering from puerperal fever. It also occurs in adults dying with pyæmia not dependent upon embolism.

Another degree is primary, *genuine desquamative pneumonia*. Its relation to the consecutive form is analogous to true Bright's disease, as compared with the renal affections occurring in some general disease. It is generally seen at an advanced period (four to six weeks or later), and is usually more fully developed in the upper parts, advancing downwards. In cases of six or eight weeks' duration, the volume and weight of the diseased lungs or lobes are considerably increased, the surface smooth, dull, covered with cuticular fibrine or united by organized adhesions. The pleura is swollen, presenting here and there ecchymoses, especially over the lower lobes. The lungs do not collapse, even on section; the elasticity is diminished or absent. The friability is increased, even to diffused softening. The cut surface shows the lobar diffuse expansion of the alteration, a more or less diminished, in parts even complete absence of air, and a feeble granulation. The last condition is due to the thickened inter-alveolar parenchyma. Considerable blood is present. The longer the process has persisted, the greater the abundance of granular pigment, whereby the tissue is slaty-gray or black. On scraping the surface, a slight amount of gelatinous fluid is obtained, bloody and opaque from the presence of numerous cell elements. Under the microscope, one finds exfoliated epithelium from the alveoli and bronchi, fatty degenerated, often containing brown or black pigment. Neither pus, mucus, nor coagula are present. The expectoration, even in the first week, is characteristic; that, too, where the symptoms might suggest croupous pneumonia. One finds abundant alveolar epithelium, with occasional ciliated epithelium. The longer the duration of the disease, the greater the abundance of the epithelium, granular and pigmented corpuscles. Free fat granules and free nuclei are also present. Later, the alveolar epithelium undergoes a myeline degeneration. The enlarged, rounded cells become granular corpuscles, the granules being dull, their outlines indistinct, the cells appearing as pale, clear bodies. The pulmonary epithelium shows nuclear proliferation; with newly formed cells of various size and shape.

In croupous pneumonia, such a degeneration occurs towards recovery. Stellate and spindle cells are also obtained from the cut surface. The pigment is often extremely abundant. The alveolar framework is thickened by the new formation of tissue.

Though this disease is generally fatal, it is not always so. Complete recovery may take place. A protracted course may be accompanied by chronic fatty degeneration of the epithelium, in one case extending over a year when death occurred, the clinical symptoms of pulmonary phthisis being present.

A second result of genuine desquamative pneumonia is cirrhosis of the lungs. In this case, the development of the pulmonary connective tissue predominates by far over the superficial epithelial alterations. The term is applied when the connective tissue appears "as fibrous cicatrices or tumors, in which the alveolar parenchyma and the minutest bronchi are enclosed, obliterated and destroyed." At times, muscular cells predominate to such a degree as to demand the term muscular cirrhosis. This connective tissue may be pigmented, or gray and translucent; in the latter instance, generally combined with the preceding form. These cirrhotic nodules may become ossified.

Cheesy pneumonia is a result of the genuine desquamative pneumonia, and is the highest degree of the same, terminating in necrosis, and is acute, subacute or chronic. The cause of the frequency of cheesy pneumonia is found in a proliferation of peri-arterial tissue, in addition to the development of embryonal tissue in the purer forms of genuine desquamative pneumonia. In the subacute and chronic forms, one finds evidence of retrograde or final processes, either a return to the normal condition in the desquamative portions or cirrhosis, chronic fatty degeneration and the formation of cavities. As peri-bronchitis is seated in the adventitia of the acartilaginous finer and finest bronchi, it deserves the term peri-bronchiolitis. There is a simple form, without the production of pus, and a purulent form accompanied by such.

Though the tubercle belongs to the lymph system, and is analogous to a lymphoid organ, it represents a peculiar sort of lymphoid formation, in that the protoplasm of its giant cells becomes firm, the substance cementing the nuclei becomes carnified, its connective tissue periphery becomes fibroid. Regarding the connective tissue cells as the forerunners of fibrillary connective tissue, and of undeveloped endothelium, a special irritament stimulates them to produce the giant cells, and hence the remaining constituents of the tubercular lymphoma. Thus the tubercle may develop from the connective tissue cells (Langhans) and the endothelium (Rindfleisch, Klebs). The tubercle may become entirely absorbed. Small tuberculous cavities may follow the cheesy degeneration of tubercles; calcification may also occur. The seat of the tubercle is always wherever connective tissue lymph vessels and small arteries, with lymphatic sheaths, are present, and therefore exists in the inner fibrous layer of the mucous membrane of the larger bronchi, in the wall of the bronchioles and in the interlobular connective tissue of the lungs, even in the walls of the alveoli. The acute miliary tubercle of the lungs is, clinically, essentially a local disease, a desquamative pneumonia, distinguished from the pure form only by the fact that giant cells appear beneath the proliferating epithelium of the alveolar walls; therewith the tubercle, from the beginning, attains the interior of the alveolus, and, later, through local infection, miliary tu-

tubercles may become developed in the swollen frame-work, provided with formative connective tissue elements. Miliary tuberculosis is always acute, advances not only from the apex downwards, but from the alveolar parenchyma towards the bronchioles. This peculiarity belongs to desquamative pneumonia also, and is equally true of the parenchymatous affection, whether tubercles are developed in it or not. Almost always an acute, punctate, cheesy, lobular pneumonia develops in the nearest alveoli; hence the larger yellow miliary tubercles of the lungs are tuberculous lymphomata, with central degeneration, surrounded by degenerating and degenerated desquamated epithelium of the neighboring alveoli. This condition is a subacute miliary tuberculosis. The chronic form is the result of the acute, when retrograde changes in the entire pulmonary parenchyma occur, in addition to the punctate caseation.

Hence, a peribronchitis nodosa is a chronic miliary tuberculosis. In fact, there is no other tuberculosis of the lungs than the acute miliary form, which may extend by numerous acute secondary growths. A secondary capillary bronchitis may occur, but not always. It may be so slight that cough is absent. If this be present, microscopic examination of the sputum will present characteristic evidence.

The theory that miliary tuberculosis is a specific disease, is based upon the fact that a cheesy deposit is almost always present in the body. This cheesy deposit originates from earlier completed inflammatory conditions. It must not be completely encapsuled, surrounded by firm fibroid tissue.

The presence of tubercles in the immediate vicinity of the nodule (infectious through vicinity), as well as the simultaneous appearance of the miliary tubercles in the various organs.

The seat of the miliary tubercles in the connective tissue containing lymph vessels.

The analogy of the histological structure with that of the normal lymphoid organs, even in the physiological condition (not functional) which calls them into life.

Tuberculosis, as an infectious disease, is not single (miliary carcinosis).

The immediate inoculability from one human being to another (not inheritance).

Tuberculosis, as a rule, does not occur in company with other infectious diseases.

Finally, the results of the inoculation of animals.

Acute miliary tuberculosis is co-existent with desquamative pneumonia, not necessarily cause or result of the inflammation. It is, locally considered, an inflammation, with the development of tubercles, and the tubercular lymphomata may be found in cheesy cellular infiltration.

Only those inflammations should be called tubercular which bear, not accidentally, but inherently and necessarily, the condition of producing tubercular lymphomata, simultaneously with the appearance of the inflammation, which, however, remains limited to the corresponding inflamed portions of tissue. Hence, of the various inflammations of the lungs, the cheesy pneumonia alone deserves the name of tubercular inflammation.

Tubercular pneumonia is primary, acute; miliary tuberculosis is

secondary, infectious. The former is a constitutional disease, hereditary, and a tuberculous inflammation may occur in almost all the tissues and organs of the body. The person with a tuberculous constitution is one whose organic activity has a tendency to respond to slight irritation by an inflammatory exudation unusually rich in cells. The irritament is constitutional, not infectious, as in miliary tuberculosis, and the tubercular pneumonia is merely an enhancement of the genuine desquamative pneumonia. This exudation must have a tendency to caseation.

Under pulmonary phthisis, is comprehended the advancing destruction of the respiratory organs, and, more particularly, the decay and emaciation of the body, caused by such an encroaching lung disease. The causes are, first and foremost, parenchymatous or desquamative pneumonia, allied to which are the peri-bronchitic inflammations.

Regarding acute miliary tuberculosis as a form of phthisis, it would be an infectious phthisis. Tuberculous pneumonia would be an inflammatory phthisis. Infectious and inflammatory phthisis are both embraced under the term pulmonary consumption, for the desquamative pneumonia is common to both. Both are distinguished from each other in that the infectious phthisis never creates destruction, while the inflammatory form seldom advances without ulceration. Pulmonary consumption is either primarily inflammatory or secondarily infectious. Only the primary, inflammatory form is constitutional; the latter runs an acute or chronic course.

The individual constitution is merely one factor in the development of phthisis; it is phthisis latent. External causes must also be present; the effect must be parenchymatous irritation and inflammation of the respiratory organs.

Catarrhal and croupous pneumonia and chronic bronchial catarrh do not lead to phthisis. Catarrhal affections of the pulmonary organs become more frequent as one leaves the tropics for the higher latitudes; at the same time, the northernmost parts of the earth possess a certain immunity from phthisis. On the contrary, the tropics show the greatest frequency and the greatest rapidity in its course. So with regard to elevation. High table lands and mountains are the homes of catarrh and bronchitis, while a height of 2000 feet above the sea level forms the limit for the occurrence of phthisis. As regards the atmospheric influence, the temperature of the air and moisture play the most important parts. Sudden, extreme and frequent changes of temperature prevent compensation on the part of the body. Hence, the temperature of the air and its rapid changes must be regarded as the occasional causes of inflammatory phthisis, and in most regions where phthisis is prevalent there is a high degree of moisture.

Friedländer's investigations are experimental, a pneumonia being produced by section of the vagi (Traube), as a result of which the buccal fluids enters the lungs. An acute inflammation of the lungs follows, with a regular typical course. Examination with the microscope, six hours after the experiment, shows a grumous or thread-like mass filling the alveoli, containing more or less white blood corpuscles; in addition, granular corpuscles, at times containing pigment. These cells differ from those arising from the bronchial wall, and are regarded as arising from the normal epithelium of the alveolar wall, these having become more granular, thicker, and finally detached. An

examination made nine hours after the experiment, shows that the fibrous wall of the alveolus remains intact; the interstitial tissue surrounding the vessels and bronchi is thickened, from the presence of numerous lymphoid cells. Upon the alveolar wall, in addition to the granulo-fibrillary material, the swollen epithelium and red blood corpuscles, are found numerous lymphoid cells, with double or multiple round nuclei. The accumulation of lymphoid cells in the bloodvessels of the affected part is extreme, and, later, the alveoli are almost completely filled with lymphoid cells, which are surrounded by a continuous alveolar epithelium. A hyperæmia and sero-hæmorrhagic exudation in the alveoli takes place, then the presence of numerous lymphoid cells in the bloodvessels, interstitial tissue and in the alveolar cavities; the epithelium of the latter first swells in the transuded serum, afterwards undergoes fatty degeneration without taking an active part in the inflammatory process. This form of pneumonia differs from the fibrinous, croupous form, but is analogous to the catarrhal pneumonia of children.

In an appendix, the author refers to the publication of Buhl, whose theory of the pathology of desquamative pneumonia differs so widely. He opposes the view of the latter with regard to the endothelial nature of the alveolar epithelium, and maintains that the same is not to be considered as endothelium, but as a direct continuation of the bronchial epithelium. He also opposes the idea that the lymphoid cells are present in the alveoli through aspiration from the bronchioles, but re-asserts that they occur primarily in the alveoli. He considers that the desquamative pneumonia of Buhl is rather the œdematous condition of the epithelium, in connection with which there is no active participation of the interstitial tissue.

